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# Do synchronous airway lesions predict treatment failure after adenotonsillectomy in children less than 3 years of age with obstructive sleep apnea?

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ABSTRACT

*Objectives:* Determine the efficacy of adenotonsillectomy and the role of synchronous airway lesions in treatment failure in children younger than 3 years of age with obstructive sleep apnea.
*Methods:* A retrospective chart review was conducted for children younger than 3 years of age with obstructive sleep apnea who were evaluated and treated at a tertiary care hospital between 2005 and 2011. All participants underwent adenotonsillectomy or powered-intracapsular tonsillectomy with adenoidectomy and had both pre- and post-operative polysomnograms. Children eligible for airway evaluation underwent flexible laryngoscopy, direct laryngoscopy or bronchoscopy. For analysis, participants were categorized as cured or not-cured with an obstructive apnea–hypopnea index (OAHI) threshold of $\geq$1.4 indicating residual obstructive sleep apnea.
*Results:* Thirty-nine children met inclusion criteria and 41% had a post-operative OAHI $\leq$ 1.4 by polysomnogram. Children failing adenotonsillectomy, (OAHI $\geq$ 1.4) had a significantly higher pre-operative OAHI ($p < 0.001$) and lower nadir $SpO_2$ ($p < 0.03$) than those considered cured. Thirty-eight percent of the total population underwent airway evaluation, and synchronous airway lesions were identified in 60% of that cohort. None of the children required surgery for their synchronous airway lesions and there was no significant difference between outcome groups in number of patients who underwent airway evaluation or had synchronous airway lesions ($p = 1$ and $p = 0.14$, respectively).
*Conclusions:* Adenotonsillectomy is effective for obstructive sleep apnea in children younger than 3 years of age and the presence of a synchronous airway lesion does not necessarily predict treatment failure.


## 1. Introduction

Obstructive sleep apnea (OSA) is characterized by prolonged incomplete or intermittent complete obstruction of the upper airway [1]. It is estimated to effect 2–3% of the pediatric population and when left untreated can result in systemic and pulmonary hypertension, ventricular remodeling, neurocognitive impairment, among other problems [1–6]. Adenotonsillectomy (AT) is considered the first-line-treatment for children with OSA and reported cure rates range from 60% to 90% [1,7–9].

The effectiveness of AT is considerably lower for children younger than 3 years, with reported cure rates ranging from 0 to 35% [10,11]. Synchronous airway lesions (SALs) have been identified in 59–67% of children <3 and are thought to be a potential cause of failure to cure. In previous studies, despite the high incidence of SALs detected, less than 4% of the studied children required surgical intervention for these SALs [12,13]. This has led to considerable debate regarding the utility of airway evaluation including direct laryngoscopy and bronchoscopy at the time of AT and whether the presence of SALs are predictive of treatment failure.

This study was designed to assess the effectiveness of AT and determine whether presence of SALs contributes to treatment failure in children <3 years of age with OSA.

## 2. Methods

This is a retrospective chart review of pediatric patients <3 years of age with OSA seen between January 1st, 2005 and December 31st, 2011 in the pediatric otolaryngology clinic at our institution. The study was approved by the institutional review board (IRB) at the Cleveland Clinic Foundation. Patients were identified using International Statistical Classification of Diseases and Related Health Problems (ICD-9) code of 327.23 and filtered by

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age. Only children who had both a pre- and post-operative polysomnogram (PSG) and underwent AT or powered-intracapsular tonsillectomy (PITA) with adenoidectomy were eligible for inclusion. Patients were excluded from analysis if they were older than 36 months at the time of surgery, had undergone prior airway surgery or had a pre-existing condition, which would otherwise require airway evaluation, such as a tracheostomy.

All patients underwent standard overnight PSG at our institution in an American Academy of Sleep Medicine accredited laboratory. Sleep studies were attended continuously by a sleep technologist and interpreted by a board-certified/eligible pediatric sleep-medicine physician. The monitored parameters included: single-lead electrocardiogram (ECG), snoring, continuous airflow with thermistor and nasal pressure transducer, chest and abdominal effort, oxygen saturation via pulse oximeter, transcutaneous $pCO_2$, and body position via video monitoring. Apnea was defined as the absence of airflow for 2-breath duration with or without a fall in oxygen saturation. Hypopnea was defined by a 50% or greater reduction in the nasal pressure or thermistor signal lasting for duration of at least 2-breaths and accompanied by a 3% or greater desaturation from pre-event baseline, or an arousal. At least 90% of the event's duration must have met the amplitude reduction criteria for hypopnea. The apneas were classified as obstructive if there was continued evidence of respiratory efforts during the event. OSA was identified by an apnea–hypopnea index (OAHI; rate of apneas + hypopneas per hour of sleep) >1.

Charts were reviewed for patient age, gender, body mass index (BMI), comorbidities and pre- and post-operative PSG data. All post-operative patients were admitted to the hospital for overnight observation. Clinical data from the perioperative period was evaluated for adverse events including laryngospasm, post-operative desaturation below 90%, post operative bleeding and dehydration requiring re-admission.

Airway evaluation by flexible nasolaryngoscopy, sleep endoscopy and/or direct laryngoscopy/bronchoscopy, was performed on select patients based on clinician judgment for patients with significantly elevated pre-operative OAHI values or suspicion for airway lesions. All sleep endoscopies were performed in pediatric operating rooms staffed by pediatric anesthesiologists under spontaneous ventilation. Board certified pediatric otolaryngologists performed all airway evaluations and operative, procedural and clinical notes were reviewed to identify SALs.

SALs, as in previous studies were defined as any partial or complete obstructive nasal, nasopharyngeal, oropharyngeal, hypopharyngeal, laryngeal, or endotracheal lesions [12,13]. As described in previous literature, subglottic narrowing was sized by amount of obstruction and graded by Cotton-Myer grading scale. Vascular compression or dynamic narrowing of trachea was considered significant when there was more than 50% narrowing of lumen visualized endoscopically. Laryngomalacia was recorded when there was inspiratory collapse of supraglottic structures as previously described. Laryngeal or tracheal edema was documented if mucosal edema was seen on endoscopy. All documented SALs with the exception of adenotonsillar hypertrophy were included for analysis.

Children were categorized as cured from OSA if the respective post-operative PSG revealed an OAHI $\leq 1.4$. All other patients were considered to have failed AT. OAHI values were rounded to the nearest whole number for categorization.

Associations between cure and possible predictors were assessed using logistic regression to provide odds ratio estimates and 95% confidence intervals. *p*-values for assessing such associations were determined from chi-square tests or Fisher's exact tests for categorical variables (including SALs), and from Wald chi-square tests for continuous variables. Associations between cure and continuous post-op factors were assessed using Wilcoxon rank sum tests. Changes from pre-op to post-op in BMI and polysonogram data were assessed using Wilcoxon signed rank tests. Analyses were performed using R version 3.0.1 (www.r-project.org).

## 3. Results

The medical charts of 243 children were reviewed. Two hundred and four patients were excluded from analysis: 169 patients did not have both pre- and post-operative PSG data, 20 did not have a diagnosis of OSA by polysomnogram, 8 patients were older than 36 months, 3 patients had a tracheostomy requiring airway visualization, 3 patients had only an adenoidectomy and 1 patient had a cleft palate repaired prior to OSA evaluation.

Of the 39 patients included in this analysis, there were 18 males (age range: 15.12–34.29 months) and 21 females (age range: 14.93–34.65 months) with a combined average age of 26.5 months at the time of surgery (Table 1). Significant medical co-morbidities

**Table 1**
Population characteristics by outcome group. No.: number, mos.: months, PSG: polysomnogram.

| Population characteristics | | | | | |
|---|---|---|---|---|---|
| Characteristic | Total | Cured; OAHI $\leq 1.4$ | Non-cured; OAHI $\geq 1.5$ | *p*-value | Odds ratio 95% CI |
| Total number of patients | 39 | 16 | 23 (59.0%) | | |
| Age at time of surgery (mos.) | 26.5 (5.7) | 27.5 (6.0) | 25.8 (5.5) | 0.35 | 0.85 (0.59–1.20)[a] |
| Gender | | | | 0.69 | 1 |
| Female | 21 | 8 | 13 (61.9%) | | 1 |
| Male | 18 | 8 | 10 (50.0%) | | 0.63 (0.13–3.01) |
| Medical comorbidity | | | | 0.041 | |
| No | 20 | 5 | 15 (75.0%) | | 1 |
| Yes | 19 | 11 | 8 (42.1%) | | 0.24 (0.06–0.95) |
| Neurologic deficit | | | | 0.045 | |
| No | 31 | 10 | 21 (67.7%) | | 1 |
| Yes | 8 | 6 | 2 (25.0%) | | 0.16 (0.03–0.93) |
| Genetic abnormality | | | | 0.15 | |
| No | 34 | 13 | 21 (61.8%) | | 1 |
| Yes | 5 | 4 | 1 (20.0%) | | 0.15 (0.01–1.19) |
| Interval between first PSG and second PSG (mos.) | 8.34 (6.94) | 7.43 (6.16) | 8.98 (7.50) | 0.49 | 1.11 (0.82–1.50)[b] |
| Interval between surgery and second PSG (mos.) | 6.81 (7.01) | 5.11 (6.21) | 7.99 (7.42) | 0.22 | 1.24 (0.88–1.75)[b] |
| Post-operative complications | | | | 0.82 | |
| No | 26 | 11 | 15 (57.7%) | | 1 |
| Yes | 13 | 5 | 8 (61.5%) | | 1.17 (0.30–4.58) |

[a] Per 3 months greater age.
[b] Per 6 months longer interval.

**Table 2**
Incidence of comorbidity by type among entire patient population; some patients had more than one comorbidity.

| Most common comorbidity by type | |
|---|---|
| Medical comorbidity | Number of patients |
| Neurologic | |
| Cerebral palsy | 3 |
| Fetal alcohol syndrome | 1 |
| Epilepsy | 3 |
| Developmental delay, not otherwise specified | 3 |
| Genetic | |
| Trisomy | 2 |
| Rubinstein–Taybi | 1 |
| 22q11 | 1 |
| Other | |
| Gastroesophageal reflux | 10 |
| Asthma | 6 |
| Pre-term birth | 5 |
| Patent ductus arteriosus | 4 |
| Dysphagia | 2 |
| Wolf-Parkinson White | 1 |
| Torticollis | 1 |
| Bronchopulmonary dysplasia | 1 |
| Chronic pancreatitis | 1 |

were observed in 19 (48.72%) patients, with 8 having neurologic and 5 having genetic abnormalities (Tables 1 and 2).

AT was the most common primary surgical intervention for OSA, comprising 76.92% of all operations. Other surgical interventions included powered-intracapsular tonsillectomy and adenoidectomy (PITA; 20.51%) and tonsillectomy with partial adenoidectomy (2.56%).

Post-operatively, the mean OAHI for all patients decreased from 21.57 to 3.33 ($p < 0.0001$; Table 3), producing an average OAHI reduction of 77.66% (Table 3). Surgical intervention also produced significant improvements in nadir oxygen saturation ($p < 0.001$), percent of sleep time with oxygen saturation below 90% ($p < 0.002$) and maximum end-tidal carbon dioxide ($p < 0.002$).

On post-operative PSG, 16 patients had an OAHI $\leq 1.4$ and 31 had an OAHI $\leq 5$. Based on post-surgical PSG, patients were categorized as either cured or not-cured as defined above. Sixteen (41%) met the criteria for cure. 35 patients out of 39 patients in the study achieved OAHI reduction >50% (Fig. 1). There were no significant differences in age at time of surgery or gender between outcome groups (Table 1). Patients who failed to achieve a cure had a significantly higher pre-operative OAHI, lower $nSpO_2$ and higher percent of sleep time with $SpO_2 < 90\%$ (Table 4). Interestingly, patients with any medical comorbidity or specifically, neurologic comorbidity exhibited a lower failure percentage than those without ($p = 0.041$, and 0.045 respectively).

Fifteen patients (38.46%) underwent 26 separate airway evaluations with 8 (53.33%) children receiving flexible laryngoscopy alone, either in the office or the operating room as a sleep endoscopy. The remaining 7 children (46.66%) underwent a direct laryngoscopy/bronchoscopy with or without a flexible laryngoscopy. In total, 28 SALs were identified in 8 (53%) of the 15 patients undergoing airway evaluation. Out of the 8 patients with SALs, 2 were classified as cured and 6 as not cured. All identified SALs are listed in Table 5. SALs observed on airway visualization in patients that failed are observed in Table 6.

There was no significant difference between outcome groups in the number of patients who underwent airway evaluation or had

**Table 3**
Pre-and post-surgical polysomnogram data for the entire patient population. BMI: body mass index, OAHI: obstructive apnea–hypopnea index.

| Population-wide results by polysomnogram | | | |
|---|---|---|---|
| | Pre-op | Post-op | *p*-value |
| Age | 25.0 (6.1) | 33.3 (7.7) | |
| BMI | 19.2 (10.3) | 17.1 (2.5) | 0.24 |
| OAHI | 21.6 (18.7) | 3.3 (3.6) | <0.001 |
| Min $O_2$% | 78.2 (9.8) | 88.7 (5.5) | <0.001 |
| % time $O_2 < 90$ | 6.7 (11.9) | 0.27 (0.65) | <0.001 |
| Max $CO_2$ | 50.9 (7.8) | 47.8 (10.4) | 0.06 |
| % time $CO_2 > 45$ | 26.2 (26.8) | 16.6 (25.9) | 0.32 |

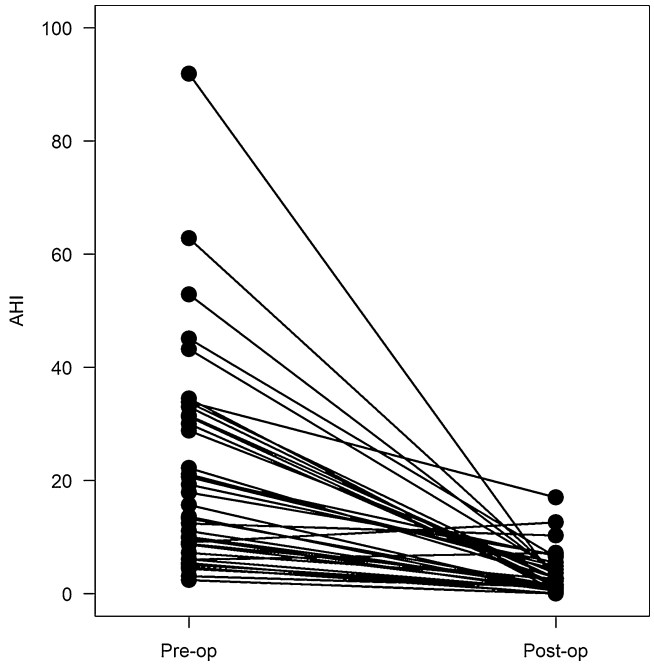


**Fig. 1.** OAHI changes before and after AT.

**Table 4**
Polysomnogram data by outcome group. BMI: body mass index, OAHI: apnea–hypopnea index.

| Comparative polysomnogram data | | | | |
|---|---|---|---|---|
| | Mean cure; OAHI $\leq 1.4$ | Mean no cure; OAHI $\geq 1.5$ | *p*-value | Odds ratio 95% CI |
| Pre-op PSG data | | | | |
| OAHI | 11.2 (9.4) | 28.8 (20.4) | 0.008 | 1.70 (1.15–2.52)[a] |
| BMI | 22.3 (15.4) | 17.1 (4.0) | 0.25 | 0.68 (0.34–1.33)[a] |
| Nadir $SpO_2$ (%) | 81.8 (7.7) | 75.7 (10.5) | 0.07 | 0.69 (0.46–1.03)[a] |
| % time $SpO_2 < 90\%$ | 2.47 (4.00) | 9.56 (14.6) | 0.09 | 1.89 (0.91–3.95)[a] |
| Max $CO_2$ | 47.9 (8.1) | 52.4 (7.4) | 0.16 | 2.46 (0.69–8.80)[b] |
| % time $CO_2 > 45$ | 18.1 (26.9) | 30.9 (26.4) | 0.23 | 1.22 (0.88–1.69)[b] |
| Post-op PSG data | | | | |
| OAHI | 0.78 (0.48) | 5.10 (3.82) | | |
| Mean OAHI change | 10.4 (9.3) | 23.7 (21.5) | 0.021 | |
| Change in nadir $SpO_2$ | 7.7 (10.8) | 10.9 (8.4) | 0.32 | |
| Nadir $SpO_2$ | 90.1 (5.0) | 87.7 (5.7) | 0.23 | |
| % time $SpO_2 < 90\%$ | 0.13 (0.27) | 0.37 (0.82) | 0.47 | |
| Max $CO_2$ | 45.3 (5.8) | 49.5 (12.5) | 0.17 | |
| % time $CO_2 > 45$ | 10.1 (21.7) | 20.4 (27.9) | 0.06 | |

[a] Per 5 units greater value of the pre-op variable.
[b] Per 10 units greater value of the pre-op variable.

**Table 5**
Synchronous airway lesions observed on airway visualization were categorized by location and are presented here.

| Identified synchronous airway lesions | | |
|---|---|---|
| Location | Lesion type | Incidence |
| Nasopharynx | Turbinate hypertrophy, hypertrophied torus tubarous, edema | 3 |
| Oropharynx | Large base of tongue, palatal collapse, lingual tonsil hypertrophy, macroglossia, lateral pharyngeal wall obstruction, elongated soft palate | 12 |
| Glottis | Omega epiglottis, laryngolmalacia, posterior epiglottis, erythema, epiglottic prolapse, laryngeal cleft, short epiglottis | 7 |
| Trachea | Tracheomalacia, vascular compression, edema, anomolous great vessels | 6 |
| Total # SALs | | 28 |

an SAL ($p = 1$, 0.1357, respectively). The observed failure rate was higher among patients with SALs (6/8 = 75%) than for patients without SALs (17/31 = 54.8%), but this was not statistically significant ($p = 0.43$). The observed incidence of SALs was higher for patients with failure (6/23 = 26.1%) compared to patients who were cured (2/16 = 12.5%). There were a total of 7 SALs in cured patients and 21 SALs in failed patients, so among patients with SALs, there was an identical average of 3.5 SALs per patient within each of the failed and cured groups (Table 7). Interestingly, 22/26 (78.57%) SALs were identified at the anatomical level of glottis or higher and none of the lower-airway lesions required surgical intervention.

Out of the 39 patients included in this study, there were 6 post-operative complications related to oxygen desaturation below 90% requiring interventions, such as oxygen supplementation and/or racemic epinephrine. One patient had a small blood clot in his nose and was extubated in the pediatric intensive care unit without difficulty. Within the 24-hour post-operative period, 1 patient experienced laryngospasm and required re-intubation. One patient was re-admitted due to dehydration and there were no complications related to post-operative bleeding.

## 4. Discussion

The goals of this retrospective chart review were to evaluate the effectiveness of AT and determine if SALs contribute to treatment failure in children <3 years of age with OSA. There is ample evidence to suggest AT is the first-line treatment for pediatric patients with OSA [1]. Previous concern over the safety of AT in children younger than 3 years of age, however, obscured the study of efficacy of this intervention in the younger pediatric population.

Recent investigations have shown that AT can reduce the post-operative OAHI to <1 in approximately 0–35% of patients [10,11].

**Table 6**
Synchronous airway lesions observed on airway visualization were categorized by location and are presented here in patients that failed.

| Identified synchronous airway lesions in patients that failed | | |
|---|---|---|
| Location | Lesion type | Incidence |
| Nasopharynx | Turbinate hypertrophy, hypertrophied torus tuberous, edema | 2 |
| Oropharynx | Large base of tongue, palatal collapse, lingual tonsil hypertrophy, macroglossia, lateral pharyngeal wall obstruction, elongated soft palate | 8 |
| Glottis | Omega epiglottis, laryngolmalacia, posterior epiglottis, erythema, epiglottic prolapse, laryngeal cleft, short epiglottis | 6 |
| Trachea | Tracheomalacia, vascular compression, edema, anomolous great vessels | 5 |
| Total # SALs | | 21 |

**Table 7**
Comparison of incidence of SALs in outcome groups.

| SAL incidence in cure vs. fail | | | |
|---|---|---|---|
| | Mean cure; OAHI ≤ 1.4 ($n = 16$) | Mean no cure; OAHI ≥ 1.5 ($n = 23$) | $p$-value |
| Patients with SALs | 2 (12.5%) | 6 (26.1%) | 0.43 |
| Patients without SALs | 14 | 17 | |
| Number of SALs | 7 | 21 | |

Mitchell and Kelley [11] defined OSA as a respiratory distress index (RDI) greater than 5, and observed a 35% cure rate after AT in children <3 years of age. Brigance et al. [10], however, defined OSA as an OAHI > 1 and found a 0% cure rate after AT in children less than 2 years of age.

Similar to Mitchell and Kelley [11] and Brigance et al. [10], the patient population in this study included patients with craniofacial abnormalities, genetic disorders and neurologic deficits. In our study, 41% of the patient population achieved an OAHI ≤ 1 after AT, and 79% achieved an OAHI of ≤5. Although a strict definition of cure suggests a low success rate, overall evaluation would suggest clinical improvement in significant portion of patients. For example, 35 patients out of the 39 patients in the study achieved OAHI reduction >50%. 31 patients out of 39 achieved post-op OAHI < 5.5. Lastly, mean OAHI value of the "no cure" changed from 28.79/h to 5.10/h postoperatively suggesting nearly most patients improve after surgery. These cure rates were surprising and impressive considering that these patients were seen in a tertiary care subspecialty practice where nearly third of the patients had a significant neurologic or genetic anomaly (Table 2). In addition, in this study, children with a comorbidity and specifically neurological comorbidity had lower failure percentage. This suggests perhaps the adenotonsillar hypertrophy and obstruction is more likely the sole, causative source of OSA in these children. However, further studies need to elucidate this relationship.

Treatment failure after AT for OSA arises from unknown factors in many instances. Recent investigations have revealed the incidence of SALs to be between 59% and 67% in children <3, suggesting multi-level airway narrowing may be a potential source of residual OSA after AT [12,13]. In 2007, Mandell and Yellon [12] reported 59% of children younger than 18 months had SALs, of which, >50% were laryngomalacia and laryngeal edema. Similarly, in 2011, Rastatatter, et al. [13] observed a 67% incidence of SALs in children undergoing AT for sleep disordered breathing. The most common SALs in that report were tracheal cobblestoning, subglottic stenosis (grade I) and trachobronchomalacia.

In the Mandell and Yellon cohort [12], no children required surgical intervention for their SALs, and <4% of the patients in the Rastatter, et al. [13] study required surgical intervention (2 subglottic cyst excisions, 1 tracheostomy and 1 posterior laryngeal cleft repair). Both reports concluded airway endoscopy is an effective diagnostic modality for SALs, but their results suggest surgery has a limited role in the management of SALs in pediatric OSA.

Consistent with these prior reports, [12,13] the incidence of SALs in our population was 53% and there were no surgical interventions required for SALs below the oropharynx. Furthermore, there was no statistically significant difference in the incidence of SALs between patients with an OAHI ≤1.4 or >1.4, suggesting SALs are not predictive of treatment outcome in younger pediatric patients with OSA.

More recent investigations have identified non-anatomical etiologies for elevated OAHIs and OSA in the pediatric population. In 2012, Wasilewska et al. [14] identified a significant 5-point

reduction in OAHI after a short trial with proton pump inhibitors for patients with both OSA and GERD. The authors concluded that a questionnaire for gastroesphageal reflux in patients with OSA might be of benefit. This comes as an addition to the known link between pediatric obesity and OSA, which was further strengthened by Nino and colleagues [15] who demonstrated an inverse relationship between abdominal adiposity and outcome in patients with OSA. These studies highlight the significant, negative impact non-structural pathology can have on OSA.

The large percentage of patients achieving a cure with an OAHI $\leq$ 1.4 (41%) (79% if cure is defined as OAHI $<$ 5) combined with the uniform incidence of SALs among outcome groups in this study lends support to a growing body of research that suggests that although commonly found in children $<$3 years o age, SALs may not be the sole source of failure to achieve cure after AT. Based on literature reviewed, non-anatomical pathologies including obesity and reflux should be investigated and addressed in cases of AT failure.

Although the primary goal of this study was not to address the post-operative complications of AT for OSA in children $<$3 years, 15.38% of patients were noted to have significant adverse events after surgery. This value is significantly higher than recent estimates and suggests caution should be used when observing patients in the post-operative period [16].

The study is limited by the small proportion of patients who underwent both pre- and post-operative PSGs, received laryngoscopies/bronchoscopies and its retrospective design. Furthermore, although all patient management was performed by 3-trained pediatric otolaryngologists, there was no uniform approach to the diagnosis, treatment, and reporting in these patients [17]. Lastly, since only about half of the patients undergoing airway evaluation had a laryngoscopy/bronchoscopy, the ability to detect subglottic and tracheal SALs is limited.

Based on the size of the study population, limitations posed by the variables listed and retrospective nature of study, it is difficult to make conclusions on the importance of performing airway evaluations in all children less than 3 years of age undergoing T&A for OSA. The intent of the study was to evaluate the incidence of SALs in these children and to see if the incidence correlates with incidence of failure to cure OSA after T&A. The results in the study are supported by findings in previous literature. Further investigations are necessary in evaluating the incidence of SALs in this population and their role in failure to cure OSA in a systematic controlled manner with uniform airway evaluation and grading mechanisms.

## Conclusions

AT is an effective treatment for OSA in children $<$3 years producing a cure rate of 41–79% depending on the criteria used to define residual disease (OAHI $\leq$ 1.4 & OAHI $\leq$ 5, respectively). Consistent with prior reports, no SALs below the level of the glottis required surgical intervention and there was no statistically significant difference in the incidence of SALs in the cured vs. failure groups. Prospective randomized studies are needed to further define the role of SALs in patients younger than 3 years of age with OSA.

## Conflict of interest

None of the authors have any conflicts of interest related to this investigation.

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## References

[1] American Academy of Pediatrics, Section on pediatric pulmonology, subcommittee on obstructive sleep apnea syndrome. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome, Pediatrics 109 (2002) 704–711.
[2] J.C. Lumeng, R.D. Chervin, Epidemiology of pediatric obstructive sleep apnea, Proc. Am. Thorac. Soc. 5 (2008) 242–252.
[3] Raouf S. Amin, Thomas R. Kimball, Judy A. Bean, Jenny L. Jeffries, J. Paul Willging, Robin T. Cotton, Sandra A. Witt, Betty J. Glascock, Steven R. Daniels, Left ventricular hypertrophy and abnormal ventricular geometry in children and adolescents with obstructive sleep apnea, Am. J. Respir. Crit. Care Med. 165 (2002) 1395–1399.
[4] L. Brunetti, S. Rana, M.L. Lospalluti, A. Pietrafesa, R. Francavilla, M. Fanelli, et al., Prevalence of obstructive sleep apnea syndrome in a cohort of 1207 children of southern Italy, Chest 120 (2001) 1930–1935.
[5] N.A. Goldstein, M. Fatima, T.F. Campbell, R.M. Rosenfeld, Child behavior and quality of life before and after tonsillectomy and adenoidectomy, Arch. Otolaryngol. Head Neck Surg. 128 (2002) 770–775.
[6] CE, R.T. Brouillette, Abnormalities of breathing control and airway maintenance in infants and children as a cause of cor pulmonale, Pediatr. Cardiol. 3 (1982) 249–256.
[7] M. Friedman, M. Wilson, H.C. Lin, H.W. Chang, Updated systematic review of tonsillectomy and adenoidectomy for treatment of pediatric obstructive sleep apnea/hypopnea syndrome, Otolaryngol. Head Neck Surg. 140 (2009) 800–808.
[8] J. Chan, J. Edman, P. Koltai, Obstructive sleep apnea in children, Am. Fam. Physician 69 (2004) 1147–1154.
[9] Rakesh Bhattacharjee, Leila Kheirandish-Gozal, Karen Spruyt, Ron B. Mitchell, Jungrak Promchiarak, Narong Simakajornboon, Athanasios G. Kaditis, Deborah Splaingard, Mark Splaingard, Lee J. Brooks, Carole L. Marcus, Sanghun Sin, Raanan Arens, Stijn L. Verhulst, David Gozal, Adenotonsillectomy outcomes in treatment of obstructive sleep apnea in children: a multicenter retrospective study, Am. J. Respir. Crit. Care Med. 182 (2010) 676–683.
[10] J.S. Brigance, R.C. Miyamoto, P. Schilt, D. Houston, J.L. Wiebke, D. Givan, et al., Surgical management of obstructive sleep apnea in infants and young toddlers, Otolaryngol. Head Neck Surg. 140 (2009) 912–916.
[11] R.B. Mitchell, J. Kelley, Outcome of adenotonsillectomy for obstructive sleep apnea in children under 3 years, Otolaryngol. Head Neck Surg. 132 (2005) 9681–9684.
[12] D. Mandell, R. Yellon, Synchronous airway lesions and esophagitis in young patients undergoing adenoidectomy, Arch. Otolaryngol. Head Neck Surg. 133 (2007) 375–380.
[13] J.C. Rastatatter, J.W. Schroeder Jr, A. French, L. Holinger, Synchronous airway lesions in children younger than age 3 years undergoing adenotonsillectomy, Otolaryngol. Head Neck Surg. 145 (2011) 309–313.
[14] J. Wasilewska, J. Semeniuk, B. Cudowska, M. Klukowski, K. Debkowska, M. Kaczmarski, Respiratory response to proton pump inhibitor treatment in children with obstructive sleep apnea syndrome and gastroesophageal reflux disease, Sleep Med. (2012) 824–830.
[15] G. Nino, M.J. Gutierrez, A. Ravindra, C.L. Nino, C.E. Rodriguez-Martinez, Abdominal adiposity correlated with adenotonsillectomy outcome in obese adolescents with severe obstructive sleep apnea, Pulm. Med. (2012) 351037 10.1155/2012/351037 Epub 2012 Nov. 18.
[16] D.J. Spencer, J.E. Jones, Complications of adenotonsillectomy in patients younger than 3 years, Arch. Otolaryngol. Head Neck Surg. 138 (4) (2012) 335–339.
[17] P.S. Roland, R.M. Rosenfeld, L.J. Brooks, N.R. Friedman, J. Jones, T.W. Kim, S. Kuhar, R.B. Mitchell, M.D. Seidman, S.H. Sheldon, S. Jones, P. Robertson, Clinical practice guideline: polysomnography for sleep disordered breathing prior to tonsillectomy in children, Otolaryngol. Head Neck Surg. 145 (2011) S1–S15.